Surgical Hemorrhage, Damage Control, and the Abdominal Compartment Syndrome

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ABSTRACT

The morbidity and mortality associated with surgical hemorrhage are considerable, particularly when relaparotomy is necessary. This complication can usually be avoided with comprehensive preoperative patient evaluation and meticulous surgical technique. The damage control sequence is a useful surgical strategy when severe intraoperative coagulopathy or hemodynamic instability is present. Abdominal compartment syndrome is a potentially lethal phenomenon that can occur following laparotomy or large-volume fluid resuscitation, or both. Early recognition and intervention are critical to survival of the patient when this syndrome occurs.

KEYWORDS: Surgical hemorrhage, damage control, abdominal compartment syndrome

Objectives: Upon completion of this article, the reader should be familiar with strategies for the prevention and management of surgical hemorrhage and abdominal compartment syndrome.

> The only weapon with which the unconscious patient can immediately retaliate upon the incompetent surgeon is hemorrhage.
>
> William Stewart Halsted, 1912

Of the many battles the surgeon encounters during years in practice, none is more demoralizing or critical than that waged against uncontrolled hemorrhage. Rapid, deliberate action is necessary to gain control of unanticipated surgical bleeding, and vigilance is essential to recognize ongoing hemorrhage postoperatively.

First, we focus on bleeding complications in abdominal and pelvic surgery and review techniques for preventing and managing intraoperative bleeding. This includes damage control surgery, which is a useful strategy to temporize bleeding in critically ill, coagulopathic patients. Finally, we discuss abdominal compartment syndrome. Although this potentially lethal phenomenon is predominantly described in the trauma literature, it can occur following fluid resuscitation and abdominal operations in patients with a variety of diagnoses.

SURGICAL BLEEDING—PREVENTION

Preoperative Evaluation

As in more traditional forms of combat, the best offense against surgical bleeding is a good defense. A serious effort should be made preoperatively to identify patients who have an elevated risk for bleeding related to abnormalities of platelet function and coagulation. This involves a detailed history. Patients should be asked about unusual bruising or bleeding. In the patients with

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known hemostatic disorders, proper evaluation and preparation are mandatory to minimize the risk of surgical bleeding.

Hemophilia A (factor VIII deficiency), hemophilia B (factor IX deficiency), and von Willebrand's disease are the most common congenital coagulation disorders. Optimal preparation of patients with these or other congenital disorders requires preoperative communication between the surgeon and the patient's hematologist. Recommendations for factor replacement and duration of perioperative therapy vary according to disease severity and the magnitude of the surgical procedure. In general, surgical procedures on patients with hemophilia A or B should be preceded by the administration of recombinant or plasma-derived monoclonal factor VIII or IX concentrates, and patients with von Willebrand's disease should receive 1-desamino-8-D-arginine vasopressin (DDAVP) or factor VIII concentrates with von Willebrand factor (vWF).[1–3]

Warfarin (Coumadin) therapy is a common cause of acquired coagulation dysfunction. Patients maintained within an international normalized ratio (INR) range of 2 to 3 can be expected to exhibit normalization of the prothrombin time/INR ~5 days after cessation of warfarin.[4] Bridging therapy with intravenous unfractionated heparin or subcutaneous low-molecular-weight heparin may be indicated for patients at a high risk for thromboembolism.[4] Preoperative consultation with the patient's cardiologist or primary care physician, or both, can be helpful in this situation. When rapid reversal of anticoagulation is necessary for emergency procedures, administration of fresh-frozen plasma with or without vitamin K is indicated.

Congenital platelet disorders are relatively rare. A variety of medical conditions and medications can induce platelet dysfunction or thrombocytopenia, or both. Antiplatelet medications, such as aspirin, nonsteroidal anti-inflammatory drugs, and clopidogrel (Plavix™), should be discontinued 10 to 14 days prior to elective surgery. DDAVP infusion and platelet transfusions are useful measures in preparation for emergency operations.[2] Platelet transfusion is contraindicated for patients with thrombotic thrombocytopenic purpura and hemolytic-uremic syndrome. These forms of microangiopathic hemolytic anemia can both be treated by total plasma exchange with cryoprecipitate supernatant plasma.[1] Close consultation with a hematologist during the perioperative period is imperative.

Maintenance of surgical hemostasis in patients with vascular and connective tissue disorders, such as Ehlers-Danlos syndrome, can be challenging for even the most technically talented surgeon. Tissue friability may also be increased in patients with prior radiation treatment, steroid use, or vitamin C deficiency (scurvy). Careful tissue handling and meticulous surgical technique must be employed to avoid catastrophic vascular injuries in this group of patients.[2]

Abnormal hemostasis should be anticipated in patients with renal failure and hepatic dysfunction. Patients with short bowel or malabsorption syndromes may have impaired coagulation secondary to vitamin K deficiency. In addition to preoperative hemodialysis, platelet dysfunction associated with uremia may be improved with DDAVP, cryoprecipitate, or conjugated estrogens.[1–3] Patients with hepatic failure should undergo thorough preoperative coagulation studies with plasma transfusions as indicated during the perioperative period. The operating surgeon should also be prepared to contend with extensive venous collaterals in patients with portal hypertension.

Intraoperative Considerations

One of the most difficult sites of surgical bleeding encountered by colon and rectal surgeons is in the presacral region. This complication may be avoided with careful surgical technique and a comprehensive understanding of the vascular anatomy in this area. Protected by the presacral fascia, the presacral venous plexus is formed by the middle sacral, lateral sacral, and communicating veins and drains into the internal iliac veins. Sacral basivertebral veins emerge from foramina located on the pelvic surface of the third to fifth segments of the sacral body, connecting the internal vertebral venous system and the external venous plexus. These veins may be large in caliber and can produce high-pressure bleeding when disrupted.[5,6]

In a clinical and anatomic study of presacral bleeding associated with abdominoperineal proctectomy, Wang et al[6] observed patterns of hemorrhage and identified four surgical maneuvers that commonly cause trauma to the presacral and basivertebral veins. Severe hemorrhage was most commonly attributed to blunt finger dissection between the posterior rectal wall and the sacral surface with violation of the presacral fascia. Sharp dissection, performed from the pelvis or upward from a perineal approach, may also result in laceration of the presacral fascia if performed too close to the sacrum. Finally, clamping and lifting bleeding vessels on the presacral surface can cause vascular injury.

Although there is no substitute for meticulous sharp dissection within the avascular presacral space, several techniques have been described for the management of severe presacral hemorrhage. Sterile thumbtacks are particularly useful in cases of sacral basivertebral venous injury, especially when the lacerated vein retracts into its sacral foramen. After digital control of bleeding has been established, a sterile tack can be pressed into the foramen for permanent hemostasis.[7] Lower pressure bleeding from the presacral venous plexus can usually be controlled with suture ligation or temporary packing.

Application of a laparoscopic stapling device can be used to tamponade bleeding veins against the bony surface of the sacrum.[5,8] Other techniques described in the literature include use of balloon devices,[9] hemostatic sponges with cyanoacrylate adhesive,[10] and electrocautery "welding" of rectus muscle to the bleeding site.[11–13]

DAMAGE CONTROL

At some point, every surgeon is confronted by severe, diffuse intraoperative bleeding that is unresponsive to conventional hemostatic measures. In these critically ill patients with massive hemorrhage, this condition evolves through the combined effects of hypothermia, metabolic acidosis, and coagulopathy. Resuscitation with large volumes of crystalloid and blood products contributes to hemodilution, thrombocytopenia, and a relative reduction in coagulation factors. Intraoperative efforts to reverse profound coagulopathy in the setting of ongoing blood loss and hypothermia can be futile.

In 1983, Stone et al[14] reported a series of 31 patients who developed major coagulopathy during laparotomy for abdominal trauma. The mortality rate of the initial 14 patients treated with standard laparotomy and definitive repair of all injuries was 93%. After an algorithm was adopted by which abbreviated laparotomy, abdominal packing, and temporary abdominal closure are followed by resuscitation and correction of coagulopathy in the intensive care unit and planned reexploration, the mortality rate decreased to 35%. This strategy, commonly referred to as the "damage control" sequence, is well represented in the trauma literature. The application of damage control principles has also been described in other surgical settings, including urologic, gynecologic, and colon and rectal surgery.[15–18]

Indications and Selection of Patients

The decision to perform damage control surgery must be made rapidly based on the patient's overall physiologic state. Indications for this approach include hemodynamic instability, coagulopathy, hypothermia, severe metabolic acidosis ($pH < 7.30$), inaccessible major venous injury, shock secondary to sepsis, and suboptimal patient response to resuscitation in the setting of a prolonged surgical procedure.[15,18,19] When operating on a critically ill patient with any of these factors, the surgeon must periodically reassess the condition of the patient intraoperatively. Communication with the anesthesia team is essential when considering early termination of a laparotomy.

The Damage Control Sequence

The damage control sequence in abdominal surgery consists of three distinct stages: initial laparotomy with temporary closure of the abdomen, secondary resuscitation, and planned reoperation with definitive organ repair. A disciplined, coordinated approach is necessary to complete the process with optimal results.

Once a surgeon has decided to employ the damage control approach, the initial laparotomy should be completed in an expeditious fashion. Evacuation of hematoma and intraperitoneal contamination should be performed first, followed by necessary organ repairs. Major vascular injuries should be controlled using clamps, suture, or shunts. Temporary aortic occlusion may be necessary for control of hemorrhage during exploration and revascularization efforts. Hollow organ injuries can be temporized with suture or stapling techniques. When intestinal resections are performed, the bowel is left in discontinuity with the ends stapled. Laparotomy pads may be packed over areas of persistent bleeding. Temporary abdominal closure is performed by reapproximating the skin edges using towel clips or suture, leaving the fascia open to minimize intra-abdominal pressure and preserve fascial integrity.[14,15,18,19] If massive visceral edema has developed as a result of fluid resuscitation, abdominal closure may not be possible. Several options, including placement of absorbable mesh, sterile intravenous bags, sterile X-ray cassette covers, and vacuum-assisted devices, are extremely useful under these circumstances.[20,21]

The goal of the second stage in the damage control sequence is the correction of the underlying coagulopathy and restoration of normal physiology. Postoperatively, the patient is transported to the intensive care unit for continued resuscitation. Core rewarming can be achieved using radiant heat lights, heating blankets, or chest tube placement with warm saline pleural lavage.[22] Administration of warm blood, crystalloid, and plasma may be performed using a rapid infusion device. Periodic laboratory assessment of electrolyte, hematology, and coagulation profiles should be obtained with aggressive correction as indicated. Mechanical ventilation and continuous hemodynamic monitoring are essential.[15,19,22]

Once rewarming, correction of coagulopathy, and optimization of fluid status and hemodynamics have been achieved, the patient is returned to the operating room for removal of packs and definitive surgery. This is usually performed between 24 and 72 hours after the initial operation. After all laparotomy pads have been removed, the peritoneal contents should be reinspected for missed injuries or nonviable intestinal segments. Restoration of intestinal continuity can be accomplished using standard anastomotic techniques, with endostomy creation if indicated. If prolonged dependence on mechanical ventilation is anticipated, placement of an enteral feeding tube should be considered. Closure of the abdominal fascia in standard fashion is performed at the completion of the operation. If the fascia cannot be

approximated without tension, placement of absorbable mesh or a vacuum-assisted closure device is recommended.[14,18,19,22–24]

The considerable morbidity and mortality associated with damage control surgery underscore the importance of selection of patients for implementation of this strategy. Common complications include wound infection, intra-abdominal abscess, wound dehiscence, bile leak, enterocutaneous fistulas, multiple system organ failure, and abdominal compartment syndrome.[14,15,23,24]

UNPLANNED RELAPAROTOMY FOR POSTOPERATIVE HEMORRHAGE

Recognition of Ongoing Bleeding

Perhaps the most difficult aspect of the management of postoperative hemorrhage is the early recognition of this potentially devastating complication. During the postoperative period, the surgeon must remain alert for signs of ongoing hemorrhage. Subtle clinical signs of blood loss may be missed or falsely attributed to postoperative fluid shifts, especially when no major bleeding complications were encountered during the initial operation. The decision to return to the operating room should be based on whether the patient's estimated postoperative blood loss exceeds the expectations of the operative surgeon.

In a series of 166 operations performed for postoperative hemorrhage in trauma patients, Hirshberg et al described direct and indirect signs of bleeding. Direct signs, consisting of bleeding from drains and other evidence of external bleeding, contributed to the decision to reoperate in 74% of patients. The diagnosis of ongoing hemorrhage in the remaining cases was made on the basis of indirect clinical signs, including hemodynamic instability, hematocrit drop, and abdominal distention. Seventy-seven percent of the abdominal reexplorations were performed within 24 hours of the primary procedure.[25]

Patterns of Postoperative Bleeding

When performing an abdominal reexploration for hemorrhage, the surgeon should be mindful of the various patterns in which postoperative bleeding can occur. Discreet sites of bleeding are typically easier to recognize and control. This type of postoperative hemorrhage usually results from incomplete hemostasis at a site of previous repair. Bleeding from a specific vessel or group of vessels can result from vascular injury during suture ligation, suture displacement from a short vascular "stump," or inadequate vascular occlusion by a suture because of disease-induced vessel wall rigidity.[26] Bleeding from iatrogenic injuries to the spleen or liver that were inadequately addressed or unrecognized at the primary operation should also be considered.[25] Once the source of bleeding is identified, standard surgical techniques for vascular repair or ligation, splenorrhaphy, or hepatorrhaphy should be employed as indicated.

Anastomotic bleeding from suture or staple lines can occur after surgical procedures requiring intestinal resection. Blood per rectum in excess of normal postoperative hematoma evacuation should alert the surgeon to this possible bleeding etiology. Although endoscopy has both diagnostic and therapeutic potential in this situation, it should be employed with great caution in the setting of a newly created intestinal anastomosis.[27]

Diffuse bleeding from raw surfaces is often the most challenging mode of bleeding to control. This condition may result from ongoing coagulopathy or unrecognized conditions of hemostatic or platelet dysfunction. Identification and correction of the underlying disorder are essential in this situation. Abdominal packing is an option if adequate hemostasis cannot be achieved.[2,25,28]

Complications of Relaparotomy

Early relaparotomy for bleeding or other postoperative complications carries a considerable amount of risk. Several studies have demonstrated an overall mortality rate between 29 and 71% for patients requiring abdominal reexploration in the early postoperative period. The mortality associated with reoperation for intraperitoneal hemorrhage (27 to 40%) is typically lower than that of laparotomies performed for infectious complications. Factors associated with increased morbidity and mortality include age older than 50 years, emergent primary laparotomy, peritonitis at the time of primary laparotomy, and the development of multiple system organ failure prior to relaparotomy.[29–32] In light of these risks, the decision to return to the operating room for abdominal reexploration should be calculated carefully.

ABDOMINAL COMPARTMENT SYNDROME

Compartment syndrome is a descriptive term for the detrimental effects of elevated pressure within a confined anatomical space. Traditional examples include tissue swelling and ischemia within the fascial compartments of the extremities and increased intracranial pressure associated with hemorrhage or closed head injury.[33] Increased pressure within the peritoneal cavity, or intra-abdominal hypertension, can have profound effects on multiple organ systems. This constellation of clinical signs is often referred to as abdominal compartment syndrome.

Predisposing Factors

Although the majority of the literature pertaining to abdominal compartment syndrome is derived from the trauma population, this condition can affect a wide

variety of patients. Spontaneous causes include massive ascites, intra-abdominal abscess, ileus, intestinal obstruction, ruptured aortic aneurysm, tension pneumoperitoneum, acute pancreatitis, pregnancy, chronic ambulatory peritoneal dialysis, and mesenteric venous thrombosis. Postoperative and iatrogenic causes include hemorrhage (intraperitoneal or retroperitoneal), visceral edema, acute gastric dilatation, abdominal closure under tension, abdominal packing, and reduction of large peritoneal or diaphragmatic hernias. Post-traumatic elevations of intra-abdominal pressure typically occur as a result of hemorrhage or postresuscitation visceral edema.[33] Secondary abdominal compartment syndrome can occur as a result of large-volume resuscitation in patients with traumatic injuries to sites other than the abdominal compartment.[34]

Assessment of Intra-abdominal Pressure

Normal intra-abdominal pressure varies with respiration and ranges between 0 and 5 mm Hg.[33,35,36] Although exact measurements vary between authors, intra-abdominal hypertension is usually defined as intra-abdominal pressure greater than 20 mm Hg.[34–38]

Intra-abdominal pressure can be measured using various direct and indirect techniques. Direct measurement requires placement of an intraperitoneal catheter connected to an external pressure transducer or saline manometer. Indirect measurements are more practical in clinical situations. These methods rely on the placement of pressure-monitoring devices into intra-abdominal organs that reflect the intra-abdominal pressure. Early studies in animal models described percutaneous catheter placement for inferior vena cava pressure monitoring.[33,37] Gastric pressure can be measured by water manometry through a nasogastric or gastrostomy tube.[33,37] The most common technique used in clinical practice is the measurement of urinary bladder pressure. After instilling 50 to 100 mL of liquid into the bladder through an indwelling transurethral catheter and clamping the distal tubing, pressure measurements can be obtained by attaching a manometer or bedside monitor to a needle inserted in the specimen collection port of the catheter. With the patient in supine position, the symphysis pubis is used as the zero point for monitor calibration.[33–36,38]

Physiologic Consequences of Intra-abdominal Hypertension

Elevated intra-abdominal pressure has an adverse effect on multiple organ systems. Abdominal distention increases the pressure within the adjacent pleural space. Pulmonary compliance is consequently impaired, as demonstrated by elevations in peak airway pressures in ventilated patients. A reduction in cardiac output occurs from the additive effects of diminished venous return and increased afterload. A subsequent decline in hepatic and mesenteric blood flow can result in visceral ischemia. Renal dysfunction with oliguria or anuria occurs secondary to decreased perfusion, compression of the renal veins, and a lower glomerular filtration rate. Although the physiologic mechanism is unclear, elevations of intracranial pressure have also been associated with intra-abdominal hypertension.[33–38]

Aggressive resuscitation with crystalloid and blood products is necessary to maintain an adequate circulatory volume, especially in the presence of ongoing hemorrhage. In the setting of intra-abdominal hypertension, profound capillary leakage occurs through the combined effects of a dilutional decrease in plasma oncotic pressure and a pressure-induced impairment of mesenteric venous and lymphatic outflow. Ascites and visceral edema develop, further increasing the intra-abdominal pressure. Once this positive feedback cycle has been activated, the progression to abdominal compartment syndrome occurs fairly rapidly.[33,34,37,39]

Diagnosis and Treatment of Abdominal Compartment Syndrome

Abdominal compartment syndrome is a lethal condition, with mortality rates reported between 25 and 75%.[33,34,40,41] Surgical decompression within the first 24 hours is associated with improved patient outcomes.[34,41] To optimize patient survival, the surgeon must maintain a high index of suspicion and be prepared to intervene promptly.

Pulmonary dysfunction and abdominal distention are often the initial clinical indications of abdominal compartment syndrome and therefore warrant intra-abdominal pressure monitoring. Patients with large-volume resuscitation requirements should also undergo periodic intra-abdominal pressure monitoring. The diagnosis of abdominal compartment syndrome can be made when intra-abdominal hypertension is accompanied by at least one of its end-organ manifestations.

The decision to perform a decompressive laparotomy should be based on the overall clinical condition of the patient instead of relying on specific intra-abdominal pressure parameters. Although strict guidelines for surgical intervention have not been established, a strategy introduced by Meldrum et al recommends decompressive laparotomy based on bladder pressure > 20 mm Hg accompanied by one of the following: peak airway pressure > 40 mL H_2O, oxygen delivery index < 600 mL O_2/min/m^2, or urine output < 0.5 mL/kg/hr.[42]

Abdominal decompression can be performed in the intensive care unit or in the operating room. Bedside laparotomy is an option for patients with high ventilatory requirements or those too unstable for transport. When possible, the procedure should be performed in the operating room with skilled nursing and anesthesia

support. Abrupt abdominal decompression can activate a process referred to as reperfusion syndrome, in which a rapid increase in cardiac output and washout of the byproducts of anaerobic metabolism can lead to circulatory collapse. The anesthesia team should anticipate this phenomenon by administering mannitol and sodium bicarbonate prior to and during the surgical procedure.[34,40] Intra-abdominal hemorrhage and ischemic viscera requiring resection can be more easily addressed in an operating room environment.

After the anesthesia team has instituted appropriate monitoring and preoperative medications, decompressive laparotomy should proceed expeditiously . In the majority of cases, a recent laparotomy has been performed and abdominal access can be quickly obtained through the midline wound. If towel clips are present from prior damage control surgery and temporary abdominal closure, the initial removal of every other clip may prevent an abrupt drop in intra-abdominal pressure.[40] In cases of secondary abdominal compartment syndrome, abdominal decompression can be performed through a generous midline incision. When the peritoneal cavity has been exposed and any residual hematoma has been evacuated, a quick but thorough exploration should be performed. Surgical repair or abdominal packing may be employed if ongoing hemorrhage is present.

To maintain abdominal decompression, the skin and fascial edges must be left open. Several options for temporary coverage of the open abdomen have been described. These options include absorbable mesh and vacuum-assisted closure devices. Delayed fascial closure may be possible after visceral edema decreases and normal physiology has been restored. If the fascia cannot be reapproximated without tension, a split-thickness skin graft may be applied when an adequate bed of granulation tissue has formed in the open abdominal wound.[20,21,34,40] Abdominal wall reconstruction can be performed 6 to 12 months later using a fascial components separation technique or prosthetic mesh.[43]

SUMMARY

Hemorrhage and abdominal compartment syndrome are two potentially catastrophic complications of abdominal surgery. Prevention of both conditions requires careful preoperative evaluation and meticulous surgical technique. Recognition of the clinical signs associated with postoperative hemorrhage and abdominal compartment syndrome requires vigilance on the part of the operating surgeon. Prompt surgical intervention is essential for survival of the patient.

DISCLOSURE

The authors have no conflicts to disclose relative to this article.

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